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Pulmonary Dysfunction in Parkinson's Disease Is Associated with Age and Systemic Inflammation Rather Than Motor Phenotype: A Cross-Sectional Study

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Abstract

Respiratory dysfunction is an underrecognized component of Parkinson's disease (PD), but its relationship with neurological disease severity, motor phenotype, and systemic inflammation remains incompletely understood. This study aimed to characterize pulmonary function in patients with PD and identify clinical and inflammatory factors independently associated with impaired spirometric performance.

Methods

This single-center observational study included 93 patients with PD. Demographic and clinical characteristics, Hoehn–Yahr stage, Unified Parkinson's Disease Rating Scale (UPDRS) score, disease duration, spirometric parameters, and laboratory markers, including C-reactive protein (CRP), albumin, and haemoglobin, were recorded. Respiratory and disease-specific outcomes were assessed using the St George's Respiratory Questionnaire, COPD Assessment Test, modified Medical Research Council dyspnoea scale, and Parkinson's Disease Questionnaire-39. Patients were categorized according to Hoehn–Yahr stage, motor phenotype, and pulmonary function status, with reduced pulmonary function defined as FEV_1 <80% of predicted. Correlation analyses and multivariable regression models were used to identify factors independently associated with FEV_1, FVC, and the FEV_1/FVC ratio.

Results

The mean age was 69.58 ± 6.93 years, and 53.8% of patients were male. Reduced pulmonary function was present in 43.0% of patients. Higher Hoehn–Yahr stage was associated with greater neurological severity, longer disease duration, and poorer PD-specific quality of life, but not with significant differences in FEV_1, FVC, FEV_1/FVC, dyspnoea, or respiratory symptom scores. Motor phenotype was not significantly associated with spirometric or quality-of-life measures. Patients with reduced pulmonary function were older and had higher CRP levels than those with preserved function. Age was inversely correlated with FEV_1 ($r = -0.318$, $p = 0.002$) and FVC ($r = -0.296$, $p = 0.004$). In multivariable models, age and CRP were independently associated with lower FEV_1 and FVC, whereas age and haemoglobin were independently associated with the FEV_1/FVC ratio.

Conclusions

Reduced pulmonary function in PD was more consistently associated with ageing and systemic inflammation than with neurological disease stage, motor phenotype, or disease duration. The absence of a clear relationship between spirometric impairment and respiratory symptoms suggests that clinically relevant pulmonary impairment may remain underrecognized. Objective respiratory assessment may therefore provide complementary clinical information in patients with PD, particularly in older individuals and those with increased systemic inflammatory burden.

1. Introduction

Parkinson's disease (PD) is one of the most common neurodegenerative disorders of later life, and its clinical and public health burden continues to increase with population ageing. Although PD is traditionally defined by its cardinal motor manifestations, including bradykinesia, rigidity, tremor, and postural instability, it is now recognized as a multisystem disorder involving multiple non-motor domains. These manifestations can substantially affect functional status, quality of life, and long-term outcomes and may, in some patients, contribute to disability independently of motor impairment [1, 2].

Respiratory dysfunction represents a relatively underrecognized component of the clinical spectrum of PD. Several mechanisms may contribute to respiratory impairment, including chest wall rigidity, altered respiratory muscle coordination, postural abnormalities, upper airway dysfunction, impaired ventilatory control, and reduced cough effectiveness. Swallowing dysfunction and impaired airway protection may further increase respiratory morbidity. Importantly, respiratory abnormalities do not necessarily present with prominent dyspnoea and may develop gradually, remaining clinically unrecognized or being attributed to ageing, frailty, reduced physical activity, or the neurological disease itself [3, 4].

Previous studies have demonstrated abnormalities in pulmonary function among patients with PD, including restrictive, obstructive, and mixed ventilatory patterns. However, the relationship between pulmonary function and neurological disease characteristics remains inconsistent. Respiratory impairment does not appear to parallel motor severity in a uniform manner, and respiratory symptoms may be absent despite measurable abnormalities in pulmonary function [5, 6]. This discrepancy is clinically relevant because symptom-based assessment alone may fail to identify patients with early or clinically unapparent respiratory impairment. Consequently, pulmonary dysfunction may remain underrecognized until more advanced respiratory or swallowing-related complications develop.

Respiratory dysfunction in PD is clinically important because impaired cough, dysphagia, and reduced airway protection may contribute to bronchopulmonary complications, including aspiration pneumonia, which remains a major cause of morbidity and mortality in advanced PD [7]. However, respiratory assessment should not be restricted to patients with overt respiratory disease or established complications. Ageing itself is associated with progressive reductions in pulmonary reserve and respiratory muscle performance and may therefore increase susceptibility to respiratory dysfunction in older patients with PD. In parallel, systemic inflammation has emerged as a potential contributor to the pathophysiology of PD and its clinical progression. C-reactive protein (CRP), a readily available marker of systemic inflammation, may therefore provide additional information when pulmonary function is evaluated in this population [8, 9].

Despite increasing recognition of respiratory involvement in PD, an important unresolved question is whether impaired pulmonary function primarily reflects neurological disease severity or represents a partially independent clinical dimension of the disease. Previous studies have largely focused on specific respiratory manifestations or individual clinical determinants, whereas the relationships among pulmonary function, ageing, systemic inflammation, motor phenotype, neurological disease severity, respiratory symptom burden, and disease-specific quality of life have been less comprehensively

evaluated. In particular, whether systemic inflammatory status is independently associated with spirometric impairment after accounting for relevant demographic and clinical characteristics remains insufficiently understood.

Therefore, this study aimed to characterize pulmonary function in patients with PD and to identify demographic, clinical, and laboratory factors independently associated with impaired spirometric performance. We specifically evaluated whether pulmonary function was associated with motor phenotype and neurological disease severity and examined the independent associations of age and systemic inflammatory status with FEV_1, FVC, and the FEV_1/FVC ratio. We also assessed whether objectively measured pulmonary impairment was reflected in respiratory symptoms, dyspnoea, or PD-specific health-related quality of life.

2. Materials and Methods

2.1. Study design and participants

This single-center retrospective observational cross-sectional study included consecutive patients with idiopathic Parkinson's disease (PD) who attended the Neurology outpatient clinic of Giresun University Faculty of Medicine between January 2023 and December 2024. Clinical, laboratory, spirometric, and questionnaire data were retrospectively retrieved from institutional electronic medical records. Spirometric measurements and respiratory questionnaire assessments had been performed during routine clinical evaluations in the Department of Chest Diseases and were available in the institutional database.

Patients were included if they had complete clinical, laboratory, spirometric, and questionnaire data. Patients with missing core study variables were excluded from the final analysis.

The inclusion criteria were: (1) age ≥ 18 years; (2) a diagnosis of idiopathic PD established according to the Movement Disorder Society (MDS) Clinical Diagnostic Criteria for Parkinson's Disease by an experienced neurologist; (3) clinically stable disease at the time of assessment; and (4) availability of technically acceptable spirometry and completed respiratory and health-related quality-of-life questionnaires.

Patients were excluded if they had active pulmonary malignancy, interstitial lung disease, previous lung surgery, neuromuscular disorders other than PD, acute respiratory infection, recent pulmonary exacerbation, inability to perform technically acceptable spirometry, severe cognitive impairment preventing questionnaire completion, missing core study data, atypical parkinsonian syndromes, or secondary parkinsonism.

All neurological examinations were performed by experienced neurologists specializing in movement disorders.

2.2. Ethical considerations

The study protocol was approved by the Ethics Committee of Giresun Training and Research Hospital (Approval No. 15; 4 September 2024). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and its subsequent amendments. As this was a retrospective study based on existing clinical records, the requirement for individual informed consent was determined in accordance with the decision of the institutional ethics committee.

2.3. Demographic and clinical characteristics

Demographic and clinical characteristics included age, sex, body mass index (BMI), smoking status, alcohol use, and the presence of major comorbidities, including cardiovascular disease, diabetes mellitus, hypertension, and dyslipidaemia.

PD duration was recorded in years. Neurological disease severity was assessed using the Hoehn–Yahr (H&Y) staging system and the Unified Parkinson's Disease Rating Scale (UPDRS) total score.

Patients were categorized according to predominant motor phenotype as either tremor-dominant or bradykinesia-dominant based on the predominant clinical features documented during neurological examination by experienced neurologists. This classification was used to investigate whether pulmonary function differed according to predominant motor phenotype.

2.4. Pulmonary function assessment

Pulmonary function was assessed using spirometry. The primary spirometric parameters were forced expiratory volume in the first second (FEV_1), forced vital capacity (FVC), and the FEV_1/FVC ratio. FEV_1 and FVC were recorded as absolute values and as percentages of predicted values.

Spirometry was performed by an experienced pulmonary function technician using an Easy on-PC spirometer (ndd Medical Technologies, Zurich, Switzerland) in accordance with the 2019 American Thoracic Society/European Respiratory Society (ATS/ERS) technical standards. The device was calibrated before each testing session, and routine quality-control procedures were performed according to the manufacturer's recommendations. At least three acceptable and reproducible manoeuvres were obtained according to ATS/ERS criteria. The highest acceptable FEV_1 and FVC values were used for analysis, and the FEV_1/FVC ratio was calculated from the corresponding spirometric measurements.

Predicted values were calculated using the reference equations incorporated into the device software, accounting for age, sex, height, and ethnicity. Spirometric measurements were obtained under stable clinical conditions before bronchodilator administration and were therefore considered baseline pre-bronchodilator measurements.

For the primary categorical analysis, patients were classified according to FEV_1 expressed as a percentage of the predicted value. An FEV_1 ≥80% of predicted was considered preserved pulmonary function, whereas an FEV_1 <80% of predicted was considered reduced pulmonary function. This threshold was prespecified for the primary group comparison.

2.5. Laboratory measurements

Routine laboratory parameters obtained during clinical assessment were retrospectively recorded from the institutional medical records. The laboratory variables included serum albumin, C-reactive protein (CRP), and haemoglobin.

CRP was considered a marker of systemic inflammation, whereas albumin and haemoglobin were included as general clinical, nutritional, and haematological indicators that might be associated with pulmonary function.

Venous blood samples were obtained after overnight fasting and analysed in the central laboratory of Giresun Training and Research Hospital using standardized automated laboratory methods.

2.6. Respiratory symptoms and health-related quality of life

Respiratory and quality-of-life assessments performed during routine clinical evaluation were retrieved from the institutional medical records. Respiratory health-related quality of life was assessed using the total score of the St George's Respiratory Questionnaire (SGRQ) [10]. Respiratory symptom burden was assessed using the COPD Assessment Test (CAT) [11], and dyspnoea severity was assessed using the modified Medical Research Council (mMRC) dyspnoea scale [12]. Parkinson's disease-specific health-related quality of life was assessed using the Parkinson's Disease Questionnaire-39 (PDQ-39), a disease-specific instrument developed to assess functioning and well-being in patients with Parkinson's disease [13, 14].

These instruments were not developed specifically for the present study. The SGRQ, CAT, mMRC dyspnoea scale, and PDQ-39 were previously published instruments, and validated Turkish versions were used [10–13]. These assessments were included to determine whether objectively measured spirometric impairment was associated with perceived respiratory symptoms, dyspnoea, respiratory health status, or broader disease-specific quality of life.

All questionnaires were administered on the same clinical assessment day as the neurological examination and spirometry by trained investigators using the respective validated Turkish versions.

2.7. Study outcomes and covariates

The primary outcomes were pulmonary function parameters, including FEV_1, FVC, and the FEV_1/FVC ratio. Reduced pulmonary function, defined as FEV_1 <80% of predicted, was also examined as a categorical outcome for the primary group comparison.

Secondary analyses evaluated associations between pulmonary function and demographic and clinical variables, including age, sex, BMI, smoking status, disease duration, H&Y stage, UPDRS total score,

motor phenotype, comorbidities, inflammatory and laboratory markers, respiratory symptom scores, and PD-specific health-related quality of life.

For multivariable analyses, age, sex, UPDRS total score, disease duration, CRP, albumin, and haemoglobin were selected *a priori* based on their clinical relevance and previously reported associations with pulmonary function.

2.8. Sample size estimation

Sample size estimation was performed using G*Power version 3.1. Assuming a two-tailed correlation analysis, an expected effect size of $r = 0.30$, a two-sided α level of 0.05, and 80% statistical power, the minimum required sample size was estimated to be 84 participants. The final study population consisted of 93 patients, exceeding the minimum required sample size.

2.9. Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 31.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using skewness and kurtosis values, with values between − 1.96 and + 1.96 considered consistent with an approximately normal distribution. Continuous variables were expressed as mean ± standard deviation (SD) and median (minimum–maximum), whereas categorical variables were presented as frequencies and percentages.

Comparisons between two independent groups were performed using the independent-samples *t*-test for continuous variables and the chi-square test for categorical variables. Comparisons involving more than two groups, including comparisons according to H&Y stage, were performed using one-way analysis of variance (ANOVA). Homogeneity of variances was assessed using Levene's test. When the overall ANOVA was statistically significant, post hoc pairwise comparisons were performed using Duncan's multiple range test.

Associations between clinical variables, pulmonary function parameters, and questionnaire scores were evaluated using Spearman's rank correlation analysis. Correlation coefficients were interpreted according to their absolute values as weak (0.00–0.29), moderate (0.30–0.69), or strong (0.70–1.00).

Multivariable linear regression models were used to identify factors independently associated with pulmonary function. Separate models were constructed for FEV_1, FVC, and the FEV_1/FVC ratio as dependent variables. Age, sex, UPDRS total score, disease duration, CRP, albumin, and haemoglobin were entered simultaneously into each model. These variables were selected *a priori* based on their clinical relevance and previously reported associations with pulmonary function. Unstandardized regression coefficients (B), standard errors (SE), standardized beta coefficients (β), *p*-values, and explained variance (R^2) were reported. Multicollinearity was assessed using variance inflation factors (VIFs), with VIF values < 5 considered indicative of no problematic multicollinearity.

All statistical tests were two-sided, and a *p*-value < 0.05 was considered statistically significant.

3. Results

3.1. Study population and baseline characteristics

A total of 93 patients with Parkinson's disease (PD) were included in the final analysis. The mean age was 69.58 ± 6.93 years, and 50 patients (53.8%) were male. Hoehn–Yahr (H&Y) stages I, II, and III accounted for 57.0%, 36.6%, and 6.5% of the cohort, respectively. Forty patients (43.0%) had a bradykinesia-dominant phenotype, whereas 53 (57.0%) had a tremor-dominant phenotype.

Reduced pulmonary function, defined as FEV_1 <80% of predicted, was identified in 40 patients (43.0%). The mean FEV_1 was 79.81 ± 18.87% of predicted, while the mean FVC was 67.27 ± 15.06% of predicted. The mean FEV_1/FVC ratio was 0.92 ± 0.07. The mean SGRQ, CAT, and mMRC scores were 27.21 ± 20.65, 10.58 ± 8.09, and 0.96 ± 1.02, respectively. The mean PDQ-39 total score was 25.20 ± 13.35.

Baseline demographic, clinical, laboratory, pulmonary function, respiratory symptom, and quality-of-life characteristics are summarized in Table 1.

Table 1
Baseline demographic, clinical, laboratory, respiratory, and questionnaire characteristics of patients with Parkinson's disease

Variable	Value
Male sex	50 (53.8)
Age < 70 years	48 (51.6)
Current/former smoking	15 (16.1)
Alcohol use	2 (2.2)
Cardiac disease	19 (20.4)
Diabetes mellitus	17 (18.3)
Hypertension	39 (41.9)
Dyslipidaemia	14 (15.1)
Hoehn-Yahr stage I / II / III	53 (57.0) / 34 (36.6) / 6 (6.5)
Bradykinesia-dominant phenotype	40 (43.0)
Reduced pulmonary function, FEV1 < 80%	40 (43.0)
Age, years	69.58 ± 6.93; 69.00 (55.00–90.00)
Body mass index, kg/m²	27.93 ± 4.60; 27.16 (18.10-42.19)
Albumin, g/L	44.75 ± 2.64; 45.05 (34.80–50.60)
C-reactive protein, mg/L	2.95 ± 3.01; 2.05 (0.08–14.02)
Haemoglobin, g/dL	13.37 ± 1.48; 13.40 (9.60–16.00)
Disease duration, years	4.26 ± 3.58; 3.00 (1.00–15.00)
UPDRS total score	17.29 ± 8.14; 16.00 (4.00–43.00)
FEV1, % predicted	79.81 ± 18.87; 81.00 (20.00-124.00)
FVC, % predicted	67.27 ± 15.06; 68.00 (30.00-101.00)
FEV1/FVC ratio	0.92 ± 0.07; 0.96 (0.72-1.00)
SGRQ total score	27.21 ± 20.65; 23.42 (0.00-77.97)
CAT score	10.58 ± 8.09; 10.00 (0.00–37.00)
mMRC dyspnoea scale	0.96 ± 1.02; 1.00 (0.00–3.00)
PDQ-39 total score	25.20 ± 13.35; 22.00 (5.00–65.00)

Data are presented as n (%) or mean ± standard deviation (SD); median (minimum–maximum).

Footnote

H&Y, Hoehn–Yahr; UPDRS, Unified Parkinson's Disease Rating Scale; FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; CRP, C-reactive protein; SGRQ, St George's Respiratory Questionnaire; CAT, COPD Assessment Test; mMRC, modified Medical Research Council; PDQ-39, Parkinson's Disease Questionnaire-39. The pulmonary function category was defined as FEV_1 <80% of predicted.

3.2. Clinical and pulmonary findings according to Hoehn–Yahr stage

Clinical, pulmonary function, and questionnaire findings according to H&Y stage are presented in Table 2.

UPDRS total scores differed significantly across H&Y stages ($p < 0.001$). Mean UPDRS scores increased from 13.85 ± 6.29 in stage I to 21.41 ± 8.42 in stage II and 24.33 ± 6.35 in stage III. Post hoc analysis showed that UPDRS scores were significantly higher in stages II and III than in stage I.

Disease duration also differed significantly across H&Y stages ($p < 0.001$), increasing progressively from 2.83 ± 2.67 years in stage I to 5.38 ± 3.16 years in stage II and 10.50 ± 4.37 years in stage III.

In contrast, pulmonary function parameters did not differ significantly according to H&Y stage. Mean FEV_1 values were 82.62 ± 18.27%, 76.50 ± 20.23%, and 73.67 ± 13.38% of predicted in stages I, II, and III, respectively ($p = 0.242$). Corresponding FVC values were 69.04 ± 14.75%, 66.18 ± 15.86%, and 57.83 ± 10.01% ($p = 0.196$). The FEV_1/FVC ratio was 0.92 ± 0.07, 0.92 ± 0.08, and 0.98 ± 0.03, respectively ($p = 0.174$).

The proportion of patients with reduced pulmonary function was 39.6% in stage I, 47.1% in stage II, and 50.0% in stage III, with no statistically significant difference between groups ($p = 0.743$).

Similarly, SGRQ, CAT, and mMRC scores did not differ significantly across H&Y stages ($p = 0.513$, $p = 0.708$, and $p = 0.490$, respectively). In contrast, PDQ-39 total scores increased **significantly with** advancing H&Y stage, from 21.42 ± 12.48 in stage I to 29.38 ± 13.24 in stage II and 35.00 ± 10.73 in stage III ($p = 0.004$).

Table 2
Clinical, pulmonary function, and questionnaire findings according to Hoehn–Yahr stage

Variable	Stage I (n = 53)	Stage II (n = 34)	Stage III (n = 6)	p value
Male sex, n (%)	29 (54.7)	17 (50.0)	4 (66.7)	0.735
Age, years	68.57 ± 7.10; 68 (55–81)	71.38 ± 6.85; 71.5 (59–90)	68.33 ± 3.93; 67.5 (64–75)	0.164
Bradykinesia-dominant phenotype, n (%)	21 (39.6)	17 (50.0)	2 (33.3)	0.561
Reduced pulmonary function, n (%)	21 (39.6)	16 (47.1)	3 (50.0)	0.743
UPDRS total score	13.85 ± 6.29; 12 (4–30)a	21.41 ± 8.42; 20.5 (10–43)b	24.33 ± 6.35; 23 (18–35)b	< 0.001
Disease duration, years	2.83 ± 2.67; 2 (1–15)a	5.38 ± 3.16; 5 (1–14)b	10.50 ± 4.37; 10.5 (5–15)c	< 0.001
FEV1, % predicted	82.62 ± 18.27; 84 (35–124)	76.50 ± 20.23; 80 (20–112)	73.67 ± 13.38; 74.5 (58–93)	0.242
FVC, % predicted	69.04 ± 14.75; 71 (31–95)	66.18 ± 15.86; 64 (30–101)	57.83 ± 10.01; 57.5 (46–72)	0.196
FEV1/FVC ratio	0.92 ± 0.07; 0.95 (0.72-1.00)	0.92 ± 0.08; 0.93 (0.76-1.00)	0.98 ± 0.03; 0.99 (0.93-1.00)	0.174
SGRQ total score	25.42 ± 19.68; 22.46 (1.75–77.97)	28.67 ± 22.76; 29.67 (0-75.09)	34.71 ± 17.02; 34.06 (12.66–57.43)	0.513
CAT score	10.15 ± 8.23; 8 (0–37)	11.47 ± 8.29; 10.5 (0–33)	9.33 ± 6.15; 9 (0–16)	0.708
mMRC dyspnoea scale	0.85 ± 0.99; 1 (0–3)	1.12 ± 1.09; 1 (0–3)	1.00 ± 0.89; 1 (0–2)	0.490
PDQ-39 total score	21.42 ± 12.48; 16 (5–61)	29.38 ± 13.24; 27.5 (12–65)	35.00 ± 10.73; 37.5 (18–49)	0.004

Data are presented as n (%) or mean ± standard deviation (SD); median (minimum–maximum). Different superscript letters indicate statistically significant post hoc differences. Comparisons were performed using one-way ANOVA for continuous variables and the chi-square test for categorical variables.

Footnote

H&Y, Hoehn–Yahr; UPDRS, Unified Parkinson's Disease Rating Scale; FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; SGRQ, St George's Respiratory Questionnaire; CAT, COPD Assessment Test; mMRC, modified Medical Research Council; PDQ-39, Parkinson's Disease Questionnaire-39.

3.3. Pulmonary function according to predominant motor phenotype

Clinical, pulmonary function, and questionnaire findings according to predominant motor phenotype are presented in Table 3.

Male sex was more frequent among patients with a bradykinesia-dominant phenotype than among those with a tremor-dominant phenotype (67.5% vs. 43.4%; $p = 0.036$). No significant differences were observed between the two phenotype groups in age, BMI, CRP concentration, UPDRS total score, or disease duration.

Reduced pulmonary function was present in 37.5% of patients with a bradykinesia-dominant phenotype and 47.2% of those with a tremor-dominant phenotype ($p = 0.471$).

Mean FEV_1 was 83.20 ± 16.63% of predicted in the bradykinesia-dominant group and 77.25 ± 20.18% in the tremor-dominant group ($p = 0.189$). Mean FVC was 70.00 ± 13.24% and 65.21 ± 16.11%, respectively ($p = 0.133$), while the FEV_1/FVC ratio was 0.91 ± 0.08 and 0.93 ± 0.07 ($p = 0.185$).

No significant differences were observed between motor phenotypes in SGRQ, CAT, mMRC, or PDQ-39 scores (all $p > 0.05$).

Table 3
Clinical, pulmonary and questionnaire findings according to predominant motor phenotype

Variable	Bradykinesia dominant (n = 40)	Tremor dominant (n = 53)	p value
Male sex, n (%)	27 (67.5)	23 (43.4)	0.036
Age, years	69.27 ± 6.60; 70 (55–79)	69.81 ± 7.23; 69 (56–90)	0.714
Body mass index, kg/m²	27.14 ± 3.97; 26.61 (19.95–36.81)	28.51 ± 4.98; 27.68 (18.10-42.19)	0.156
C-reactive protein, mg/L	2.87 ± 3.58; 1.20 (0.10-14.02)	3.00 ± 2.60; 2.12 (0.08-13.00)	0.884
UPDRS total score	18.90 ± 8.87; 18 (5–43)	16.08 ± 7.40; 14 (4–32)	0.098
Disease duration, years	5.05 ± 4.20; 4 (1–15)	3.66 ± 2.94; 3 (1–13)	0.074
Reduced pulmonary function, n (%)	15 (37.5)	25 (47.2)	0.471
FEV1, % predicted	83.20 ± 16.63; 81.50 (56–124)	77.25 ± 20.18; 81 (20–112)	0.189
FVC, % predicted	70.00 ± 13.24; 71 (46–101)	65.21 ± 16.11; 65 (30–91)	0.133
FEV1/FVC ratio	0.91 ± 0.08; 0.93 (0.72-1.00)	0.93 ± 0.07; 0.96 (0.76-1.00)	0.185
SGRQ total score	27.04 ± 18.98; 23.12 (0-77.97)	27.34 ± 22.01; 24.71 (1.05–75.09)	0.945
CAT score	10.17 ± 7.60; 8.50 (0–33)	10.89 ± 8.50; 10 (0–37)	0.677
mMRC dyspnoea scale	0.92 ± 1.02; 1 (0–3)	0.98 ± 1.03; 1 (0–3)	0.794
PDQ-39 total score	25.70 ± 13.33; 23 (9–65)	24.83 ± 13.48; 21 (5–62)	0.758

Data are presented as n (%) or mean ± SD; median (minimum-maximum). Independent-samples t-test or chi-square test was used as appropriate.

Footnote

BMI, body mass index; CRP, C-reactive protein; UPDRS, Unified Parkinson's Disease Rating Scale; FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; SGRQ, St George's Respiratory Questionnaire; CAT, COPD Assessment Test; mMRC, modified Medical Research Council; PDQ-39, Parkinson's Disease Questionnaire-39.

3.4. Clinical and laboratory characteristics according to pulmonary function status

Patients were categorized according to preserved pulmonary function (FEV_1 ≥80% of predicted; *n* = 53) or reduced pulmonary function (FEV_1 <80% of predicted; *n* = 40). The corresponding clinical, laboratory, pulmonary function, and questionnaire findings are presented in Table 4.

Patients with reduced pulmonary function were significantly older than those with preserved pulmonary function (71.75 ± 6.95 vs. 67.94 ± 6.52 years; *p* = 0.008). CRP concentrations were also significantly higher in the reduced pulmonary function group (3.96 ± 3.92 vs. 2.25 ± 1.95 mg/L; *p* = 0.039).

Albumin concentrations were lower in patients with reduced pulmonary function than in those with preserved pulmonary function, although the difference was not statistically significant (44.19 ± 2.43 vs. 45.14 ± 2.73 g/L; *p* = 0.092). Haemoglobin concentrations did not differ significantly between the groups (*p* = 0.656). Similarly, UPDRS total score and disease duration were not significantly different between patients with preserved and reduced pulmonary function (*p* = 0.155 and *p* = 0.831, respectively).

As expected from the predefined classification, FEV_1 was substantially lower in the reduced pulmonary function group than in the preserved pulmonary function group (62.93 ± 13.17% vs. 92.55 ± 10.74% of predicted; *p* < 0.001). FVC was also lower in the reduced pulmonary function group (55.55 ± 12.36% vs. 76.11 ± 10.05%; *p* < 0.001), as was the FEV_1/FVC ratio (0.90 ± 0.09 vs. 0.94 ± 0.05; *p* = 0.010).

Despite the differences in spirometric parameters, SGRQ, CAT, mMRC, and PDQ-39 scores did not differ significantly between the two pulmonary function groups (all *p* > 0.05).

Table 4
Clinical, laboratory, pulmonary and questionnaire findings according to pulmonary function status

Variable	Preserved pulmonary function, FEV1 > = 80% (n = 53)	Reduced pulmonary function, FEV1 < 80% (n = 40)	p value
Age, years	67.94 ± 6.52; 68 (55–80)	71.75 ± 6.95; 72 (59–90)	0.008
Body mass index, kg/m²	27.27 ± 4.11; 27.06 (19.95–42.19)	28.79 ± 5.11; 27.88 (18.10-41.62)	0.116
Albumin, g/L	45.14 ± 2.73; 45.70 (38.00-50.60)	44.19 ± 2.43; 44.50 (34.80–47.90)	0.092
C-reactive protein, mg/L	2.25 ± 1.95; 1.60 (0.08–6.19)	3.96 ± 3.92; 2.93 (0.53–14.02)	0.039
Haemoglobin, g/dL	13.43 ± 1.58; 13.30 (9.60–16.00)	13.29 ± 1.36; 13.40 (11.10–15.50)	0.656
UPDRS total score	16.25 ± 8.20; 14 (4–40)	18.68 ± 7.95; 18 (6–43)	0.155
Disease duration, years	4.19 ± 3.69; 3 (1–15)	4.35 ± 3.48; 3.5 (1–15)	0.831
FEV1, % predicted	92.55 ± 10.74; 89 (80–124)	62.93 ± 13.17; 68 (20–78)	< 0.001
FVC, % predicted	76.11 ± 10.05; 75 (61–101)	55.55 ± 12.36; 55 (30–85)	< 0.001
FEV1/FVC ratio	0.94 ± 0.05; 0.96 (0.80-1.00)	0.90 ± 0.09; 0.92 (0.72-1.00)	0.010
SGRQ total score	28.61 ± 20.85; 25.51 (1.05–77.97)	25.34 ± 20.51; 22.78 (0-75.09)	0.453
CAT score	10.21 ± 8.88; 8 (0–37)	11.08 ± 6.99; 11.5 (0–27)	0.612
mMRC dyspnoea scale	0.83 ± 1.05; 0 (0–3)	1.13 ± 0.97; 1 (0–3)	0.169
PDQ-39 total score	24.66 ± 12.59; 20 (5–61)	25.93 ± 14.43; 22.5 (10–65)	0.654

Data are presented as mean ± standard deviation (SD); median (minimum–maximum).

Footnote

CRP, C-reactive protein; BMI, body mass index; UPDRS, Unified Parkinson's Disease Rating Scale; FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; SGRQ, St George's Respiratory Questionnaire; CAT, COPD Assessment Test; mMRC, modified Medical Research Council; PDQ-39, Parkinson's Disease Questionnaire-39. Reduced pulmonary function was defined as FEV_1 <80% of predicted.

3.5. Correlations between clinical variables and pulmonary function

Spearman correlation analyses are summarized in Table 5.

UPDRS total score showed weak inverse correlations with FEV_1 (r=-0.225, p = 0.030) and FVC (r=-0.227, p = 0.029). H&Y stage was not significantly correlated with FEV_1, FVC, FEV_1/FVC, or reduced pulmonary function.

Age was inversely correlated with FEV_1 (r=-0.318, p = 0.002) and FVC (r=-0.296, p = 0.004), and was positively associated with reduced pulmonary function (r = 0.269, p = 0.009). Disease duration was not significantly associated with any pulmonary function outcome.

Among laboratory variables, CRP was weakly inversely correlated with FEV_1 (r=-0.270, p = 0.048). Albumin showed a weak positive correlation with FVC (r = 0.211, p = 0.045) and a borderline inverse association with reduced pulmonary function (r=-0.207, p = 0.050). Haemoglobin was inversely correlated with the FEV_1/FVC ratio (r=-0.246, p = 0.017).

Hypertension was positively associated with reduced pulmonary function (r = 0.230, p = 0.027), whereas dyslipidaemia was positively correlated with the FEV_1/FVC ratio (r = 0.242, p = 0.020).

No significant correlations were observed between SGRQ, CAT, or mMRC scores and FEV_1, FVC, FEV_1/FVC, or reduced pulmonary function. PDQ-39 total score was also not significantly correlated with the spirometric outcomes.

In contrast, respiratory questionnaire scores were associated with neurological disease burden: SGRQ, CAT, and mMRC scores showed positive correlations with UPDRS total score, while PDQ-39 total score showed a strong positive correlation with UPDRS total score and a moderate positive correlation with H&Y stage.

Table 5
Spearman correlations between clinical variables and pulmonary function outcomes

Variable	FEV1, r (p)	FVC, r (p)	FEV1/FVC, r (p)	Reduced PF, r (p)
Hoehn-Yahr stage	-0.198 (0.057)	-0.194 (0.063)	0.104 (0.319)	0.080 (0.446)
UPDRS total score	-0.225 (0.030)*	-0.227 (0.029)*	0.085 (0.421)	0.168 (0.108)
Disease duration	-0.125 (0.231)	-0.166 (0.113)	0.117 (0.266)	0.055 (0.601)
Age	-0.318 (0.002)*	-0.296 (0.004)*	-0.116 (0.266)	0.269 (0.009)*
Hypertension	-0.165 (0.114)	-0.169 (0.106)	0.009 (0.935)	0.230 (0.027)*
Dyslipidaemia	-0.133 (0.203)	-0.167 (0.110)	0.242 (0.020)*	0.181 (0.083)
Albumin	0.151 (0.155)	0.211 (0.045)*	-0.127 (0.234)	-0.207 (0.050)*
C-reactive protein	-0.270 (0.048)*	-0.245 (0.074)	-0.161 (0.244)	0.252 (0.067)
Haemoglobin	0.079 (0.449)	0.197 (0.059)	-0.246 (0.017)*	-0.062 (0.553)
SGRQ total score	0.077 (0.461)	0.071 (0.499)	0.060 (0.570)	-0.085 (0.416)
CAT score	-0.159 (0.128)	-0.126 (0.228)	0.058 (0.584)	0.127 (0.224)
mMRC dyspnoea scale	-0.175 (0.094)	-0.102 (0.330)	0.003 (0.977)	0.171 (0.102)
PDQ-39 total score	-0.076 (0.468)	-0.158 (0.129)	0.202 (0.052)	0.018 (0.865)

**p < 0.05. PF, pulmonary function. Reduced PF was defined as FEV1 < 80% of predicted.*

Footnote

H&Y, Hoehn–Yahr; UPDRS, Unified Parkinson's Disease Rating Scale; FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity; CRP, C-reactive protein; SGRQ, St George's Respiratory Questionnaire; CAT, COPD Assessment Test; mMRC, modified Medical Research Council; PDQ-39, Parkinson's Disease Questionnaire-39. Reduced pulmonary function was defined as FEV_1 <80% of predicted.

3.6. Multivariable analysis of factors independently associated with pulmonary function

Multivariable linear regression models were constructed separately for FEV_1, FVC, and the FEV_1/FVC ratio. Each model simultaneously included age, sex, UPDRS total score, disease duration, CRP, albumin, and haemoglobin. The complete regression results are presented in Table 6.

For FEV_1, both age and CRP were independently associated with lower FEV_1. Age was inversely associated with FEV_1 (B=-0.67, SE = 0.26, β=-0.25, p = 0.011), while CRP was also inversely associated

with FEV_1 (B=-1.84, SE = 0.70, β=-0.31, p = 0.009). The model explained 15.7% of the variance in FEV_1 (R^2=0.157). Sex, UPDRS total score, disease duration, albumin, and haemoglobin were not independently associated with FEV_1.

For FVC, age and CRP were similarly independently associated with lower FVC. Age was inversely associated with FVC (B=-0.51, SE = 0.21, β=-0.24, p = 0.013), and CRP was also inversely associated with FVC (B=-1.39, SE = 0.55, β=-0.29, p = 0.011). The model explained 18.3% of the variance in FVC (R^2=0.183). Sex, UPDRS total score, disease duration, albumin, and haemoglobin were not independently associated with FVC.

For the FEV_1/FVC ratio, age and haemoglobin were independently associated with the outcome. Age was inversely associated with the FEV_1/FVC ratio (β=-0.22, p = 0.019), as was haemoglobin (β=-0.23, p = 0.015). The model explained 17.8% of the variance in the FEV_1/FVC ratio (R^2=0.178). Sex, UPDRS total score, disease duration, CRP, and albumin were not independently associated with the FEV_1/FVC ratio.

Overall, age was independently associated with lower FEV_1, FVC, and FEV_1/FVC, whereas CRP was independently associated with lower FEV_1 and FVC. Haemoglobin was independently associated with the FEV_1/FVC ratio. Neurological disease severity, disease duration, sex, and albumin were not independently associated with pulmonary function parameters after adjustment.

Table 6
Multivariable linear regression models for pulmonary function parameters

Dependent variable	Independent variable	B	SE	β	p value	R^2
FEV1 (%)	Sex	0.81	3.63	0.02	0.823	0.157
	Age	**-0.67**	**0.26**	**-0.25**	**0.011**	
	UPDRS total score	-0.01	0.22	0.00	0.964	
	Disease duration	-0.05	0.51	-0.01	0.925	
	C-reactive protein	**-1.84**	**0.70**	**-0.31**	**0.009**	
	Albumin	-0.07	0.70	-0.01	0.927	
	Haemoglobin	0.08	1.23	0.01	0.947	
FVC (%)	Sex	-0.18	2.84	-0.01	0.950	0.183
	Age	**-0.51**	**0.21**	**-0.24**	**0.013**	
	UPDRS total score	0.13	0.18	0.07	0.475	
	Disease duration	-0.44	0.40	-0.11	0.269	
	C-reactive protein	**-1.39**	**0.55**	**-0.29**	**0.011**	
	Albumin	0.30	0.55	0.05	0.580	
	Haemoglobin	1.48	0.96	0.15	0.124	
FEV1/FVC	Sex	0.02	0.01	0.11	0.236	0.178
	Age	**0.00**	**0.00**	**-0.22**	**0.019**	
	UPDRS total score	0.00	0.00	-0.08	0.428	
	Disease duration	0.00	0.00	0.15	0.123	
	C-reactive protein	0.00	0.00	-0.16	0.172	
	Albumin	0.00	0.00	-0.10	0.297	
	Haemoglobin	**-0.01**	**0.01**	**-0.23**	**0.015**	

B, unstandardised regression coefficient; SE, standard error; β, standardised regression coefficient. The models included sex, age, UPDRS total score, disease duration, C-reactive protein, albumin, and haemoglobin. Significant predictors are highlighted in bold.

Footnote

CRP, C-reactive protein; UPDRS, Unified Parkinson's Disease Rating Scale; FEV_1, forced expiratory volume in the first second; FVC, forced vital capacity. Each model simultaneously included sex, age, UPDRS total

score, disease duration, CRP, albumin, and haemoglobin. R^2 represents the proportion of variance explained by the corresponding multivariable model.

4. Discussion

In this single-center observational study of patients with Parkinson's disease (PD), reduced pulmonary function was relatively common, with 43.0% of patients having an FEV_1 below 80% of predicted. Patients with reduced pulmonary function were older and had higher CRP concentrations than those with preserved pulmonary function. In multivariable analyses, age and CRP were independently associated with lower FEV_1 and FVC, whereas age and haemoglobin were independently associated with the FEV_1/FVC ratio. In contrast, Hoehn–Yahr stage, motor phenotype, disease duration, sex, and UPDRS total score were not independently associated with the spirometric outcomes. Furthermore, spirometric impairment was not significantly associated with SGRQ, CAT, mMRC, or PDQ-39 scores. Taken together, these findings suggest that pulmonary impairment in PD may not simply represent a manifestation of advanced neurological disease but may also be associated with ageing and systemic inflammatory burden.

Respiratory dysfunction in PD is increasingly recognized as a heterogeneous clinical problem involving multiple potentially interacting mechanisms. These include respiratory muscle dysfunction, altered chest wall mechanics, postural abnormalities, upper airway dysfunction, impaired cough and swallowing, and abnormalities in central ventilatory control [15, 16]. Such mechanisms may occur at different stages of the disease and may not progress in parallel. Consequently, respiratory impairment in PD cannot necessarily be characterized by a single physiological pattern or assumed to correspond directly to neurological disease severity.

The present findings are consistent with this broader concept. Although FEV_1 and FVC tended to be lower in patients with more advanced H&Y stages, these differences did not reach statistical significance. Similarly, the proportion of patients with reduced pulmonary function did not differ significantly across H&Y stages. Motor phenotype was also not significantly associated with FEV_1, FVC, FEV_1/FVC, or respiratory symptom scores. These findings suggest that conventional measures of neurological severity may not adequately capture respiratory impairment in PD. This interpretation is compatible with previous work indicating that respiratory abnormalities can occur even in patients with relatively mild PD and that abnormalities in ventilatory control may be present despite relatively preserved conventional pulmonary and inspiratory muscle measurements [17, 18]. Thus, the absence of advanced neurological disease should not necessarily be interpreted as evidence of preserved respiratory function.

Age was one of the most consistent factors associated with pulmonary function in the present cohort. Older age was associated with lower FEV_1 and FVC in correlation analyses and remained independently associated with both parameters after adjustment for sex, UPDRS score, disease duration, CRP, albumin, and haemoglobin. Age was also independently associated with the FEV_1/FVC ratio. This finding is

biologically plausible because ageing is accompanied by progressive changes in lung elastic properties, respiratory muscle performance, chest wall mechanics, and physiological reserve. Importantly, longitudinal studies in healthy populations demonstrate substantial interindividual variability in the rate of pulmonary function decline, indicating that chronological ageing does not affect all individuals uniformly [19]. In PD, age-related reduction in respiratory reserve may coexist with impaired mobility, postural abnormalities, swallowing dysfunction, frailty, and comorbidity, potentially increasing susceptibility to respiratory impairment [20]. Therefore, the association of age with pulmonary function in this cohort may reflect the cumulative reduction in physiological reserve that becomes clinically relevant in patients with a neurodegenerative disorder.

The association between systemic inflammation and pulmonary function represents another important finding. CRP concentrations were significantly higher among patients with reduced pulmonary function and were inversely correlated with FEV_1. More importantly, CRP remained independently associated with lower FEV_1 and FVC after adjustment for demographic, neurological, and laboratory variables. These findings suggest that systemic inflammatory burden may be clinically relevant to pulmonary function in PD. Previous studies have investigated peripheral inflammatory markers in PD, although findings have been heterogeneous and the biological pathways remain incompletely defined [21, 22].

The present study does not establish a causal relationship between inflammation and pulmonary impairment. CRP is a nonspecific inflammatory marker and may be influenced by multiple conditions, including metabolic disease, vascular comorbidity, infection, tissue injury, and other systemic inflammatory processes. Accordingly, the observed association should be interpreted as an epidemiological and clinical signal rather than evidence that CRP directly causes pulmonary dysfunction. The directionality of the association also cannot be determined from this cross-sectional design. Higher systemic inflammation may contribute to impaired pulmonary function, impaired pulmonary function may be associated with greater inflammatory activity, or both may reflect a common underlying process [23]. Nevertheless, the persistence of the CRP association after adjustment for several clinically relevant variables supports further investigation of systemic inflammatory burden as a potential correlate of respiratory dysfunction in PD.

The spirometric findings themselves also warrant careful interpretation. FVC was relatively reduced in the cohort, whereas the mean FEV_1/FVC ratio was comparatively preserved. This pattern may be compatible with reduced lung volumes rather than classical airflow obstruction; however, spirometry alone cannot establish a restrictive ventilatory defect. Complete pulmonary function testing, including lung volumes and diffusing capacity, together with respiratory muscle strength measurements, would be required to characterize the underlying physiological mechanism. In PD, reduced FVC may reflect true restriction, impaired chest wall expansion, respiratory muscle weakness, postural limitation, impaired coordination, or suboptimal inspiratory or expiratory effort. Therefore, the present results should be interpreted as evidence of reduced spirometric performance rather than as definitive evidence of a specific ventilatory defect.

An important finding was the lack of a clear relationship between spirometric impairment and respiratory symptom burden. Patients classified as having reduced pulmonary function did not have significantly different SGRQ, CAT, or mMRC scores from those with preserved pulmonary function. Similarly, respiratory questionnaire scores were not significantly correlated with FEV_1, FVC, or the FEV_1/FVC ratio. This apparent discrepancy between objective pulmonary function and patient-reported respiratory symptoms may be particularly relevant in PD. Respiratory symptoms can be subtle and may be attributed to ageing, fatigue, reduced mobility, anxiety, or neurological disease rather than recognized as manifestations of respiratory dysfunction. Qualitative studies have also shown that patients with PD may describe respiratory symptoms in heterogeneous ways, often relating them to daily activities, posture, fatigue, and functional limitations rather than identifying them specifically as pulmonary symptoms [24]. Furthermore, recent patient-reported research has distinguished dyspnoea from dystussia, suggesting that breathlessness and impaired cough represent partially distinct clinical manifestations [25]. Conventional respiratory questionnaires may therefore not fully capture the range of respiratory abnormalities occurring in PD [26].

The relationship between neurological burden and quality of life further supports the distinction between respiratory and neurological domains. In the present study, PDQ-39 scores were significantly worse with increasing H&Y stage, and PDQ-39 was strongly correlated with UPDRS total score. SGRQ, CAT, and mMRC scores were also associated with measures of neurological disease burden. However, these relationships did not translate into significant associations between spirometric impairment and disease-specific quality of life. This suggests that quality of life in PD is influenced by multiple domains and that neurological disability and pulmonary impairment may contribute to the overall clinical state through partly independent pathways. Importantly, the absence of an association between spirometry and questionnaire-based quality of life should not be interpreted as evidence that pulmonary dysfunction is clinically irrelevant; rather, it indicates that these instruments may capture different dimensions of disease burden.

The clinical implications of these findings are relevant to routine neurological assessment. Respiratory evaluation should not necessarily be limited to patients with advanced H&Y stage or prominent dyspnoea. Older patients and those with evidence of systemic inflammatory burden may represent clinically relevant groups in whom objective pulmonary assessment could be considered, particularly when respiratory symptoms, functional decline, swallowing difficulties, or impaired cough are present. Spirometry is relatively accessible and may provide information that is not apparent from neurological examination or symptom questionnaires alone. However, spirometry should be regarded as an initial assessment rather than a comprehensive evaluation of respiratory function. When clinically indicated, additional assessment of respiratory muscle strength, cough effectiveness, swallowing function, exercise capacity, or complete pulmonary function may provide complementary information. Prospective studies linking impaired lung function with clinical outcomes in PD further support the potential importance of recognizing respiratory abnormalities as part of the broader clinical phenotype [27]. Previous interventional studies have also suggested that respiratory-focused and integrative exercise approaches may improve selected respiratory outcomes in patients with PD [28, 29].

The present findings also have implications for the interpretation of respiratory impairment in PD. A respiratory abnormality should not automatically be considered a consequence of advanced motor disease. Instead, the respiratory phenotype may reflect the interaction of neurological dysfunction with ageing, systemic inflammation, reduced physiological reserve, comorbid disease, and other factors. This multidimensional interpretation may help explain why neither H&Y stage nor motor phenotype was independently associated with spirometric performance in our cohort. It may also help explain why respiratory questionnaires did not consistently identify patients with objectively reduced pulmonary function.

Several strengths of this study should be acknowledged. First, the study integrated neurological characteristics, spirometric parameters, systemic inflammatory markers, respiratory symptoms, and PD-specific quality of life within the same cohort. This allowed pulmonary function to be evaluated in relation to several clinically relevant domains rather than as an isolated physiological measurement. Second, the study included both continuous spirometric outcomes and a predefined categorical measure of reduced pulmonary function. Third, multivariable models simultaneously accounted for demographic, neurological, inflammatory, and laboratory variables, allowing assessment of factors independently associated with FEV_1, FVC, and the FEV_1/FVC ratio.

Several limitations should also be considered. First, the retrospective, single-center, cross-sectional design precludes causal inference and limits the generalizability of the findings. Longitudinal studies are required to determine whether the observed associations with age and CRP are associated with subsequent decline in pulmonary function. Second, the sample size was relatively modest, particularly for subgroup analyses according to H&Y stage and motor phenotype. Third, spirometry was performed before bronchodilator administration, and complete lung volume measurements, diffusing capacity, and respiratory muscle strength assessments were not available. Therefore, the underlying physiological basis of the observed reduction in FEV_1 and FVC could not be definitively characterized. Fourth, CRP is a nonspecific marker and cannot distinguish neuroinflammatory activity from other systemic inflammatory or comorbid conditions. Fifth, the use of an FEV_1 <80% predicted threshold for the categorical classification of reduced pulmonary function may not capture the full spectrum of abnormal spirometry and should not be considered a substitute for contemporary lower-limit-of-normal-based interpretation. Finally, residual confounding cannot be excluded despite multivariable adjustment.

Despite these limitations, the study provides clinically relevant evidence that reduced pulmonary function is relatively common among patients with PD and that its relationship with neurological disease severity is not straightforward. The consistent associations of age with FEV_1 and FVC and of CRP with both parameters suggest that ageing and systemic inflammatory burden may be relevant components of the respiratory phenotype of PD. Prospective longitudinal studies incorporating complete pulmonary function testing, respiratory muscle assessment, swallowing and cough evaluation, and serial inflammatory measurements are warranted to determine whether these factors are associated with subsequent decline in pulmonary function and clinically relevant respiratory outcomes in patients with PD.

Conclusion

In summary, reduced pulmonary function was common in this cohort of patients with Parkinson's disease and was most strongly associated with older age and higher C-reactive protein (CRP) concentrations. Although greater neurological disease severity was associated with poorer disease-specific quality of life and higher respiratory symptom burden, these clinical measures did not independently explain spirometric impairment. These findings suggest that pulmonary function in Parkinson's disease may reflect the combined contribution of neurological disease, ageing, systemic inflammation, and reduced physiological reserve. Routine respiratory assessment, particularly in older patients and those with evidence of systemic inflammatory burden, may facilitate the early identification of clinically relevant respiratory impairment. Future prospective longitudinal studies incorporating comprehensive pulmonary function testing, respiratory muscle assessment, and evaluation of swallowing and cough function are warranted to determine whether early detection and targeted management of respiratory impairment can improve clinical outcomes in Parkinson's disease.

Abbreviations

PD
Parkinson's disease
H&Y
Hoehn–Yahr
UPDRS
Unified Parkinson's Disease Rating Scale
FEV_1
forced expiratory volume in the first second
FVC
forced vital capacity
CRP
C-reactive protein
SGRQ
St George's Respiratory Questionnaire
CAT
COPD Assessment Test
mMRC
modified Medical Research Council
PDQ-39
Parkinson's Disease Questionnaire-39
ATS/ERS
American Thoracic Society/European Respiratory Society
BMI
body mass index

SD
standard deviation.

Declarations

Ethics approval and consent to participate

The study protocol was approved by the Ethics Committee of Giresun Training and Research Hospital (Approval No. 15; 4 September 2024). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and its subsequent amendments. As this was a retrospective study based on existing clinical records, the requirement for individual informed consent was determined in accordance with the decision of the institutional ethics committee.

Consent for publication

Not applicable. The manuscript does not contain identifiable individual patient data or images.

Availability of data and materials

The datasets generated and/or analysed during the current study are not publicly available due to ethical and privacy restrictions related to individual patient data but are available from the corresponding author on reasonable request and with permission from the relevant institutional ethics committee.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

R.C. conceived and designed the study, contributed to data collection, performed the statistical analysis, interpreted the findings, and drafted the manuscript. H.A.D. contributed to neurological assessment, clinical data collection, and interpretation of the findings. R.C. and H.A.D. critically reviewed and revised the manuscript. Both authors approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

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